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REPORT NUMBER

AFAMRL TR-80-73

AD-A092714

TITLE (and Subtitle)

EVALUATION OF ERG AS A MODEL OF CEREBRAL TISSUE
PO₂ CHANGES WITH ENVIRONMENTAL STRESS

5. TYPE OF REPORT & PERIOD COVERED

Proceedings Report

6. PERFORMING ORG. REPORT NUMBER

8. CONTRACT OR GRANT NUMBER(s)

F33615-79-C-0500

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AREA & WORK UNIT NUMBERS

62202F

7231-18-04

11. CONTROLLING OFFICE NAME AND ADDRESS

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Command, Wright-Patterson Air Force Base, OH 45433

12. REPORT DATE

11 July 1980

13. NUMBER OF PAGES

8

14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office)

15. SECURITY CLASS. (of this report)

Unclassified

15a DECLASSIFICATION/DOWNGRADING
SCHEDULE

16. DISTRIBUTION STATEMENT (of this Report)

Approved for public release; distribution unlimited

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

18. SUPPLEMENTARY NOTES

Presented at Oxygen Transport to Tissue Satellite Symposium, XXVIIIth
International Congress of Physiological Sciences, Budapest, Hungary,
8-13 July 1980.

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

ERG

Correlation

Altitude

PO₂

Non-Invasive

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

AD A092714

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Introduction

The amplitude and latency of the a- and b-waves of the electroretinogram (ERG) and cerebral oxygenation (PO_2) were measured in the rhesus macaque as a function of hypoxic and stagnant hypoxia caused by increasing altitude (decreasing ambient PO_2) and $G_{0.5-2}$ (centrifugal force induced by a centrifuge), respectively. Previous studies had shown a decrease in the amplitude of the ERG b-wave in response to anoxia or retinal ischemia. The a-wave was relatively unaffected. The present study was designed to determine the relationship between cerebral PO_2 and ERG parameters under two types of hypoxic conditions. As an extension of the central nervous system (CNS), the retina may serve as a physiological correlate of cerebral tissue PO_2 . Good correlation of one or more ERG metrics with cerebral oxygenation would allow the use of a non-invasive technique to monitor cerebral PO_2 .

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Methods

Six M. mulatta were used as subjects. The monkeys had been previously implanted with a chronic stainless steel cranial fixture located extradurally over the left parietal lobe. A calibrated polarographic electrode (100 μ active tip) was inserted 12 mm into the parietal lobe. Recordings were made with a differential O_2 analyzer.

The ERG was recorded non-corneally using silver-silver chloride electrodes.

The active electrode was placed on the lower lid, beneath the pupil, the reference on the ipsilateral ear, and the ground at the lateral canthus (see Fig. 1).

The potentials were amplified and recorded on analog tape for analysis.

Visual stimuli were produced with a projector equipped with an electromagnetic shutter, operated at 2 Hz (see Fig. 1). Stimulus duration was 10 msec.

A translucent dome was placed over the monkey's head to diffuse the light and thereby minimize the effects of eye movements. Luminance, measured inside the dome, was 110×10^{-3} lumens/cm².

The monkey was seated in a custom-built primate chair for both the altitude and G_z procedures. ERG, ECG, and PO_2 electrodes were attached under ketamine anesthesia. Three hours were allowed for recovery before data collection began.

In the altitude chamber two control series of 64 stimuli were recorded at 0 ft. Recordings were then made at 5000, 10,000 and 2000 ft. intervals up to 30,000 ft. or until the animal lost consciousness, whichever came first. Recordings for the descending series were made at 5000 ft. intervals with a final control series at 0 ft. PO_2 readings were made before ERG recordings began. Stagnant Hypoxia recordings were made at 2.5, 3.0, 3.5, 4.0, 4.5, and 5.0 G_z . with 2.5 and 3.0, 3.5 and 4.0 or 4.5 and 5.0 recorded on a given day, with the lower G_z first. A 1.0 G_z control condition preceded and followed each G_z exposure. PO_2 readings were made at 10 sec intervals throughout the 60 sec exposure at each G_z level. ERG recordings were made during the final 35 sec.

Data Reduction and Analysis

ERG data were input to a computer of average transients (Nicolet) which summed the 64 potentials and plotted the results (see Fig. 2). The first 250 msec were chosen for analysis. Amplitude and latency were determined from the plots, with amplitude measured from the isoline. Difference scores were calculated by subtracting the amplitude and latency and PO_2 recorded at 0 ft. altitude or $1.0 G_z$ from those obtained at higher altitudes or G_z . Percent difference scores were computed by dividing the difference score by 0 ft. or $1.0 G_z$ scores. ERG waves with 0 amplitude were excluded from the analysis. Due to potentiating effects of sequential G exposures, only those data collected at 2.5, 3.5, and $4.5 G_z$ were selected for analysis. PO_2 readings tended to asymptote during the final 40 sec at a given G_z level and a mean of the four readings was used for analysis.

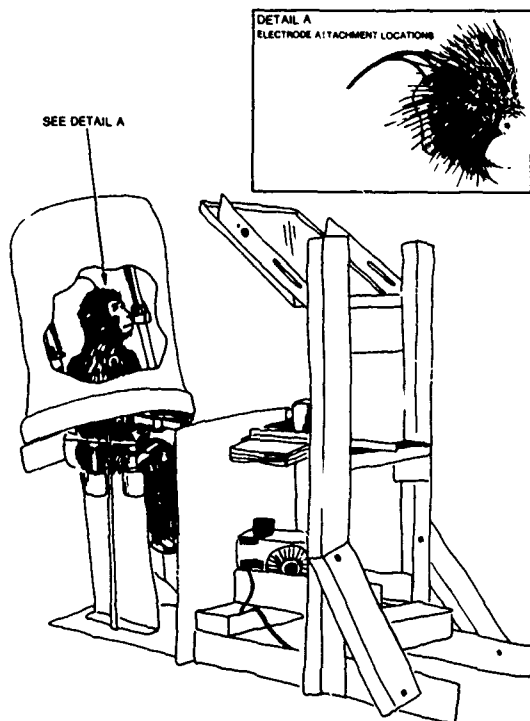


Figure 1

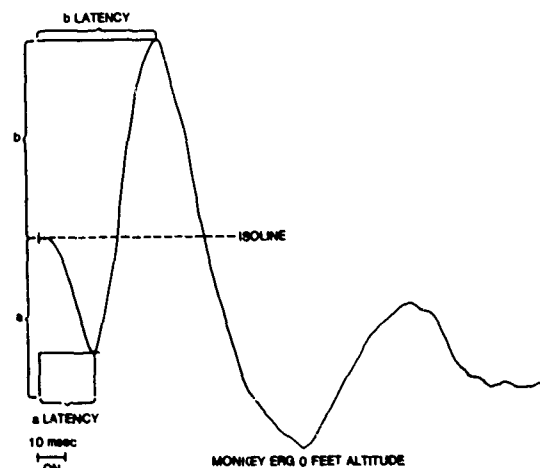


Figure 2

Results

Hypoxic hypoxia. The results from the ascending altitude series showed a decrease in a- and b-wave amplitude and an increase in the latency of both waves with decreasing ambient PO_2 . The change in b-wave latency was the largest effect and is shown in Fig. 3. A regression analysis was performed to determine which of the ERG metrics was the best predictor of cerebral PO_2 . Results of the analysis indicated that changes in PO_2 were best predicted by a quadratic equation of $\Delta \sqrt{\text{b-wave latency}}$ ($r = .71$) (see Fig. 4). Changes in PO_2 were predicted better than absolute levels, and the results of the ascending series were predicted better than the descending series. Amplitude of the a- and b-waves was not highly correlated with changes in PO_2 ($r < .35$).

Stagnant hypoxia. Percent difference scores were used for the data collected on the centrifuge. As in the altitude data, the amplitude of the ERG decreased and the latency increased with decreased cerebral PO_2 . In contrast to the altitude results, b-wave amplitude and a-wave latency were most responsive to the treatment (see Fig. 5.). A correlation analysis of the percent difference scores showed a significant decrease in b-wave amplitude ($r = .54$) and an increase in a-wave latency ($r = -.66$) with decreasing cerebral PO_2 . Amplitude of the a-wave and b-wave latency were not significantly correlated with percent change in PO_2 . A linear regression analysis was performed using percent change in b-wave amplitude and a-wave latency to predict cerebral PO_2 ($r = .78$).

Table 1 shows the results of the regression analysis. It can be seen from these data that small decreases in PO_2 are predicted by increased b-wave amplitude and shorter a-wave latencies. This "enhancement" effect was also observed at low levels of hypoxic hypoxia, particularly in b-wave amplitude. Greater decreases in cerebral PO_2 , however, are predicted by decreased b-wave amplitude and increased a-wave latency.

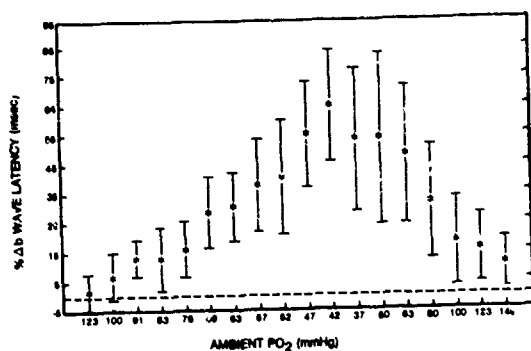


Figure 3. Percent Change in b-Wave Latency as a Function of Ambient PO_2 (Altitude). Means and 95% Confidence Intervals

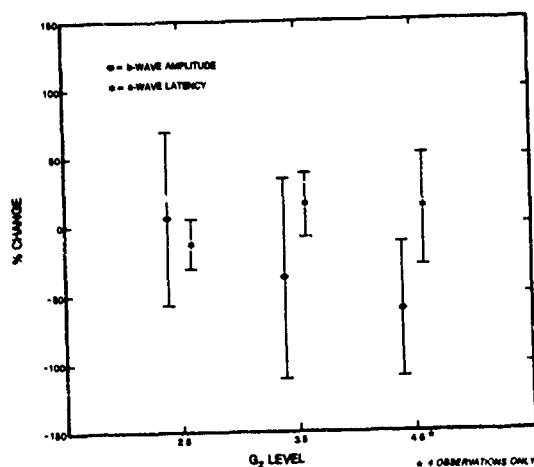


Figure 5.

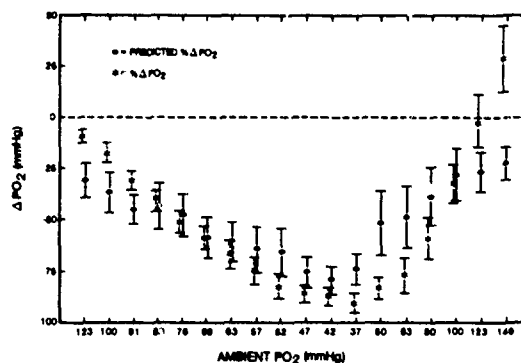


Figure 4. Actual and Predicted Percent Change in PO_2 as a Function of Ambient PO_2 (Altitude). Predicted Values are from a Regression Analysis using Percent Change in b-Wave Latency. Means and 95% Confidence Intervals

Table 1

Predicted Mean % Change in PO_2 as a Function of % Change in b-Wave Amplitude and a-Wave Latency

		% Change in b-Wave Amplitude			
		-100	-50	0	50
% Change in a-Wave Latency	~30		-25	-15	-5
	0	-50	-40	-30	-21
	30	-65	-55	-45	
	60	-80			

Note: Standard deviation = $\pm 22\%$

Discussion

The results of the hypoxic hypoxia study are in contrast to previously reported results from anoxia and retinal ischemia (Noell, 1951; Eysel, 1978). Changes in b-wave latency, not amplitude, were highly correlated with changes in cerebral PO_2 . The poor correlation with amplitude is probably due to a large "enhancement" effect that occurred between 123 and 63 mm Hg ambient PO_2 (22.8 to 7.9 mm Hg cerebral PO_2). In the stagnant hypoxia study, b-wave amplitude showed a smaller enhancement effect and was, therefore, more linearly related to changes in PO_2 . The more gradual onset of the hypoxic as opposed to the stagnant hypoxia may have contributed to the large initial increase in b-wave amplitude.

For all ERG metrics the effects of G_z were more pronounced than the effects of altitude. In particular, the a-wave was more affected by stagnant than hypoxic hypoxia. This result may be due to the relatively low oxygen consumption of the photoreceptors (0.8 vol. %. Eysel, 1978) as opposed to the rest of the retina (5.4 vol. %). The ischemia produced by G_z does not merely reduce the oxygen concentration; it may entirely cut off the blood flow to the peripheral and central retina. At high G_z levels (4.5) the ERG was not recordable, indicating a lack of retinal perfusion. Total loss of the ERG in the altitude chamber was unusual.

The results of the two studies indicate that the ERG can be used as a valid measure of changes in cerebral PO_2 caused by either stagnant or hypoxic hypoxia. The correlation coefficients could be improved by reducing the variability in the ERG recording through either on-line recording or filtering of movement artifacts.

The difference between the data obtained in the altitude chamber, with the gradual change in ambient PO_2 , and that obtained on the centrifuge, with rapid onset of ischemia, suggest that a study of the time course of ERG changes with

different rates of hypoxia onset would be of interest in describing the relationship between the ERG and cerebral PO_2 .

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Noell, W.K. Site of asphyxial block in mammalian retinae. J. Appl. Physiol. 3, 489-500 (1951).